the abscess wall and of the escape of infectious fluid into the abdomen which will set up a general peritonitis. Postpuerperal infections ure found, as a rule, in one or both broad ligaments. Those in the broad ligaments can be most satisfactorily opened extraperitoneally through a gridiron incision just above Poupart's ligament. Such accumulations should rarely, if ever, be opened through the vaginal vault.

THERAPEUTICS

UNDER THE CHARGE OF

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Clinical Studies on the Respiration. IV. The Vital Capacity of the Lungs and its Relation to Dyspnea.—This paper by PEABODY and WENTWORTH (*Arch. Int. Med.*, 1917, xx, 443) is really a continuation of the preceding in which they showed that patients with heart disease become dyspneic more easily than did healthy subjects because of their inability to increase the depth of breathing in a normal manner. The inability to breathe deeply corresponds to a diminished vital capacity of the lungs as measured by the volume of the greatest possible expiration after the deepest inspiration. The investigators employed two different methods of measuring the vital capacity. One was by use of a calibrated recording spirometer, the other by the use of an ordinary portable spirometer. They determined that the position of the patient made no practical difference in his vital capacity. One hundred and forty healthy adults were studied. They were classified according to sex and height. Of the males there were three groups: Group 1 consisted of those persons 6 feet tall or over whose normal capacity was 5100 c.c. Group 2 consisted of men between 5 feet 8.5 inches and 6 feet tall with a normal vital capacity of 4800 c.c. Group 3 consisted of men whose height was between 5 feet 8.5 inches and 5 feet 3 inches, whose vital capacity was 4000 c.c. Though the vital capacity of some subjects was very much greater than that called for by their group position, only 1 of the 96 normal males had a vital capacity more than 10 per cent. below the appropriate normal standard. Peabody and Wentworth conclude that healthy males almost invariably have a vital capacity of 90 per cent. or more of the normal standard. The women were also divided into three groups: Group 1 measured over 5 feet 6 inches; vital capacity 3275 c.c. Group 2 measured from 5 feet 4 inches to 5 feet 6 inches, with an average vital capacity of 3050 c.c. Group 3 made up of persons who measured from 5 feet 1 inch to 5 feet 4 inches; vital capacity 2825 c.c. In five of these women the vital capacity was more than 10 per cent. below the appropriate normal, but in no case was it more than 15 per cent. below. 224 observations on 124 patients suffering from cardiac disease showed that the clinical condition of the patient, more especially the tendency to dyspnea varied directly with the degree to which the vital capacity was diminished.

Patients with a vital capacity of 90 per cent. or more of the appropriate normal standard adopted for their sex and height had little or no abnormal tendency to dyspnea. Patients with a vital capacity of from 70 to 90 per cent. of the normal became short of breath on unusual exertion and must lead a restricted life, although many of them can do light work. Patients with a vital capacity of from 40 to 70 per cent. of the normal become dyspneic on moderate or slight exertion, are rarely able to work, and frequently suffer from cardiac decompensation. Those with vital capacity of less than 40 per cent. are decompensated patients, usually confined to bed, and the mortality in this group is high. There is a close correspondence in the individual case between changes of vital capacity and variations in the tendency to dyspnea. In stages of decompensation the vital capacity falls, and with recovery the vital capacity rises. Comparatively slight changes in the patient's condition may manifest themselves in changes in vital capacity. Indeed it is possible by studying the variations in vital capacity to throw much light on the course and even on the prognosis of the disease. In various other diseases in which mechanical conditions interfere with the movement of the lungs, such as pleural effusion, cirrhosis of the liver with ascites, etc., the tendency to dyspnea corresponds closely to the decrease in vital capacity. This, however, is not true for the anemias, where the dyspnea appears to be due to the low hemoglobin content of the blood. In acute nephritis the vital capacity was not decreased below the normal. In chronic nephritis without evidence of heart disease and without history of dyspnea, vital capacity was usually within the normal limits. In cardiorenal cases where dyspnea was a prominent symptom, vital capacity was usually decreased in proportion to the intensity of the dyspnea. In diseases in which dyspnea is not a prominent symptom, such as acute and chronic arthritis, diabetes, hemiplegia, tabes, and in surgical convalescence the vital capacity is usually within the normal limits, although general weakness and old age may cause a slight decrease.

Clinical Studies on the Respiration. V. The Basal Metabolism and the Minute-volume of the Respiration of Patients with Cardiac Disease.—PEABODY, WENTWORTH and BARKER (*Arch. Int. Med.*, 1917, xx, 468) report their observations on the gaseous metabolism and pulmonary ventilation of two groups of patients with heart disease. Group 1 consists of subjects in good or fairly good condition, in whom the vital capacity of the lungs was over 60 per cent. of the normal. Group 2 consists of much more severely affected patients in whom the vital capacity was 60 per cent. of the normal or less. The basal metabolism calculated from the oxygen consumption per square meter of body surface averaged 2.5 per cent. above normal in Group 1, and 12.8 per cent. above normal in Group 2. The average volume per respiration was less in Group 2 than in Group 1, and the average rate of respiration was higher in Group 2 than in Group 1. The minute-volume of air breathed averaged approximately 30 per cent. higher in the members of Group 2 than it did in those of Group 1. The relation is pointed out between the increase of the minute-volume of the more seriously affected patients and the decrease in the vital capacity of the lungs. Finally it is shown that this high minute-volume is a factor in the production of dyspnea in persons with severe heart disease.